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Perspective

Reactive Uveitis, Retinal Vasculitis and Scleritis as Ocular End-Stage of Acanthamoeba Keratitis

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ABOUT THE STUDY

Acanthamoeba Keratitis (AK) is a progressive, sight-threatening disease, occurring mostly in contact lens wearers. It is reported about 33 cases within one million contact lens wearers in the United States, which means an occurrence of about 150 new cases there, per year. In Germany, with about 80 million inhabitants, about 150 new cases have been reported in a 10-year-period. Expression of mannosilated glycoproteins on corneal epithelial cell surface is upregulated in contact lens wearers. This plays an important role in AK pathogenesis [1]. The acanthamoeba throphozoite binds to these proteins though its mannose-binding site in order to release the so-called mannose-induced protease 133 and acanthamoeba Plasminogen Activator (aPA).

Patients with Acanthamoeba keratitis experience tears and ocular pain in the early stages of the disease. In comparison to the patient's significant discomfort, the ophthalmologists note a rather modest ophthalmological state at this time. At this stage, a pseudodendriti form epitheliopathy, sometimes known as "dirty epithelium," can be seen, with spot-like multifocal stromal infiltrates and radial perineuritis [2]. A Wessely immune ring appears around the diseased area a few days later. A substantial stromal infiltration and hypopyon may be seen in cases of bacterial or mycotic coinfection. Secondary glaucoma, iris atrophy, mature cataract, scleritis, and chorioretinitis may develop in later stages.

Between 1975 and 2013, there were only eight cases of acanthamoeba extracorneal invasion reported in the literature. Scleral invasion has been described in three of these instances, while acanthamoeba sclerokeratitis has been recorded in five others. In their case series, Iovieno et al. found that 18.5 percent of patients had sclerokeratitis, with deteriorated necrotic cysts in scleral nodule biopsy. Sclerokeratitis is a T-cell-mediated immunological response that necessitates systemic immunosuppression, according to the researchers [3]. Antigens from Acanthamoeba trigger an immunological response that results in the formation of T cell clones. These T cell clones then cross-react with antigens found in the normal eye, potentially resulting in the formation of more T cell clones in a process known as "epitope spreading."

Before enucleation, one patient had a retinal artery blockage while the other had a retinal vein occlusion. These vessels have lymphocytic infiltration, according to histopathology. This could point to a local immune-mediated vasculitis with thrombosis and blockage as a subsequent symptom. We believe that peripheral vasculitis is more likely to be caused by reactive inflammation than by the acanthamoeba. This might have happened in the same way in the three patients who were reported.

In clinically relevant quantities, polyhexamethilen-biguanide and propamidin-isethionat may be cytotoxic to human corneal cells. It has also been proposed that posterior segment inflammation is linked to the toxicity of topical treatments used in AK, although earlier investigations have found that AK patients with long-term topical treatment do not have posterior pole inflammation, contradicting this notion. Nonetheless, the toxicity of biguanides may be linked to the development of mature cataracts in both cases [4]. By changing lipid membranes, destroying lens fibres, and generating electrolyte imbalance, they can disturb the lens surface, cause lenticular oxidative or osmotic stress, and lead to cataract formation.

Even if acanthamoeba trophozoites or cysts do not survive, long-term recalcitrant acanthamoeba keratitis, uveitis, retinal vasculitis, and scleritis can occur, resulting in blindness [5]. The cause of these inflammatory problems is unknown, however molecular mimicry or type III immune reactions could be to blame. As a result, systemic immune suppression may be required for a longer period of time in the late stages of acanthamoeba keratitis.

REFERENCES

1. Bloemendal H, De Jong W, Jaenicke R, Lubsen NH, Slingsby C, Tardieu A. Ageing and vision: structure, stability and function of lens crystalline. Prog Biophys Mol Biol. 2004;86(2):407-485.

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2. De Jong WW, Caspers GJ, Leunissen JA. Genealogy of the α-crystallin small heat-shock-protein superfamily. Int J Biol Macromol. 2004;22(5):151-162.
3. Klemenz R, Frohli E, Aoyama A, Hoffmann S, Simpson RJ, Moritz RL. Alpha B-crystallin accumulation is a specific response to Ha-ras and v-mos oncogene expression in mouse NIH-3T3-fibroblasts. J Mol Cell Biol. 1991;11(2):803-812.
4. Kumar SP, Udupa P, Murugesan R, Sharma KK. Significance of interactions of low molecular weight crystallin fragments in lens aging and cataract formation. J Biol Chem. 2008; 283(3):8477-8485.
5. Paron I, Scaloni A, Prescott A, Damante G, Tell G. A proteomic approach to identify early molecular targets of oxidative stress in human epithelial lens cells. Biochem J. 2004; 378(1):929-937.